

## Chapter 3

### MATES II Monitoring Program

The monitoring portion of MATES-II was designed to measure numerous air toxic compounds at different locations in the Basin in order to establish a baseline of existing air toxic ambient concentrations, as well as risk level data, and to assist in the assessment of modeling performance accuracy. Ten sites were selected and air samples were collected for up to one year.

#### 3.1 Site Location and Monitoring

With the input from ATSTRG and the Environmental Justice Task Force, ten MATES-II sites were selected. Five were selected to provide continuity with the ARB long-term trend sites (Los Angeles, Burbank, Long Beach, Rubidoux and Upland/Fontana). The Pico Rivera site was selected because monitoring equipment was available from the EPA-sponsored PAMS program. Anaheim was chosen for geographic equity, such that at least one site existed in each of the four counties. Wilmington, Compton, and Huntington Park were new sites selected to examine environmental justice concerns. Because the fixed site locations are based on EPA guidelines for “neighborhood scale” monitoring, each of these sites may also be representative of adjacent communities. Table 3-1 and Figure 3-1 shows the location of the monitoring sites.

**Table 3-1**  
**MATES II Sites**

Abbr.	Site	Period of Record	Address
<b>Fixed MATES II Sites</b>			
AN	Anaheim	4/29/98 – 3/25/99	1010 S. Harbor Blvd., Anaheim
BU	Burbank	4/05/98 – 3/31/99	228 W. Palm Ave., Burbank
CP	Compton	7/22/98 – 3/31/99	720 N. Bullis Rd., Compton
FO	Fontana	4/05/98 – 3/31/99	14360 Arrow Highway, Fontana
HP	Huntington Park	6/10/98 – 3/31/99	6301 S. Santa Fe Ave., Huntington Park
LB	Long Beach	4/05/98 – 3/31/99	3648 N. Long Beach Blvd., Long Beach
LA	Los Angeles	4/05/98 – 3/31/99	1630 N. Main St., Los Angeles
PR	Pico Rivera	4/23/98 – 3/31/99	3713 B-San Gabriel River Parkway, Pico Rivera
RU	Rubidoux	5/29/98 – 3/31/99	5888 Mission Blvd., Rubidoux
WI	Wilmington	7/31/98 – 3/31/99	900 E. Lomita Blvd., Wilmington

At each site, sampling equipment included particulate samplers; canisters; and carbonyl samplers, as well as equipment to measure key meteorological parameters. At the five long-term trend sites, monitors also measured ozone, oxides of nitrogen, and carbon monoxide. The ARB and AQMD shared the responsibility of performing laboratory analyses for VOCs and particulates. All polycyclic aromatic hydrocarbon (PAH) samples were analyzed by ARB; all carbon analyses (elemental and organic) were conducted by AQMD. Appendix III-A contains a detailed description of the analytical methods used.

Sampled compounds are listed in Tables 3-2 and 3-3. The first table shows those compounds that were measured on a routine basis, while Table 3-3 depicts those compounds that were

sampled on a more limited basis, if their presence were suspected from nearby sources. (A careful screening process was established to determine the listings in Tables 3-2 and 3-3. Unit risk factors, prior data, and analytical capabilities were considered from an original candidate list which included all state and federally designated toxic air contaminants. A subgroup of the ATSTRG was formed to consider these factors, from which the final lists were derived.)

The sampling schedule followed the USEPA National Air Monitoring schedule for particulate matter which is once every six days for a twenty-four hour period from midnight to midnight.

## 3.2 Findings

Results of the data focus on the following key topics:

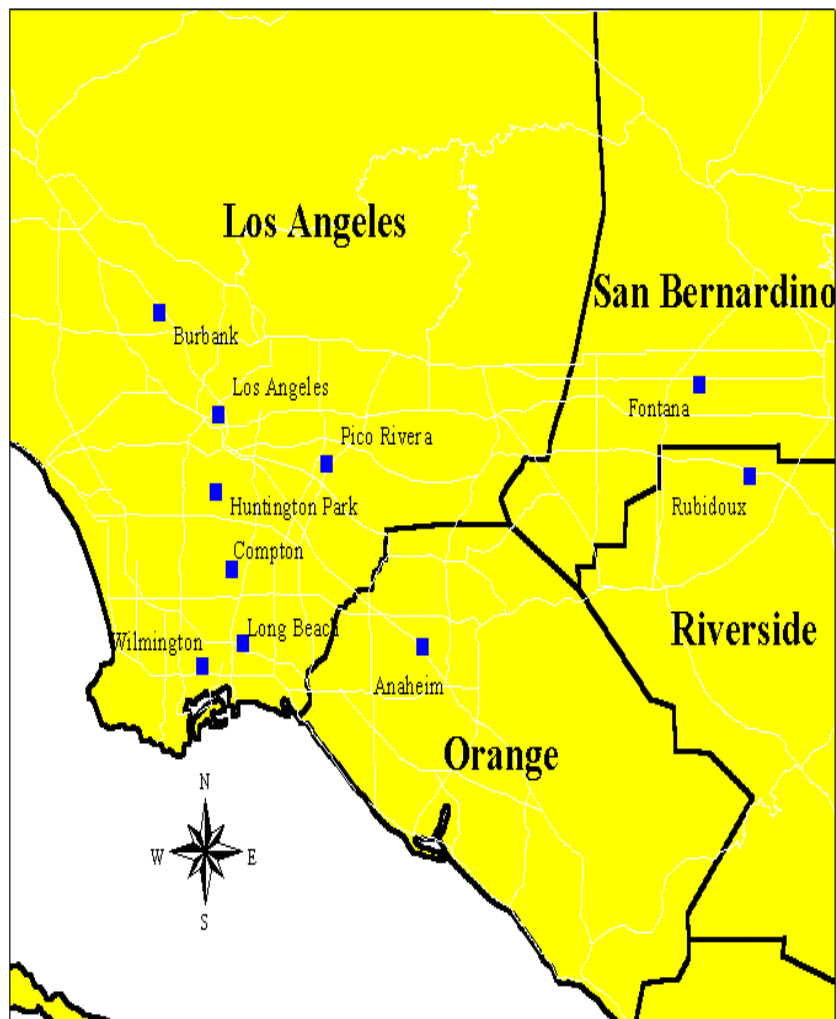
- intersite comparison of ambient toxic concentrations and cancer risks among the ten fixed sites,
- seasonal variability of ambient toxic concentrations, and
- comparison of species and source apportionment of cancer risk among the sites.

**Table 3-2**  
**Routinely Measured Compounds**

CAS No.	Chemical Name	CAS No.	Chemical Name
71432	Benzene	50000	Formaldehyde
7440439	1,3 Butadiene	75070	Acetaldehyde
106467	Dichlorobenzene (ortho- & para)		Acetone
75014	Vinyl Chloride	7440382	Arsenic
10414	Ethyl Benzene		Chromium
	Toluene	7439921	Lead
	Xylene (m-, p-, o-)	7440020	Nickel
	Styrene		Cobalt
56235	Carbon Tetrachloride		Copper
67663	Chloroform		Manganese
75343	Dichloroethane [1,1]	7723140	Phosphorous
	Dichloroethylene [1,1]		Selenium
75092	Methylene Chloride		Silica
127184	Perchloroethylene		Silver
79016	Trichloroethylene		Zinc
74783	Chloromethane		PAHs
			Elemental & Organic Carbon

**Table 3-3**  
**Compounds Considered for Measurement Based on Local Factors**

Compound	Compound
Acrylonitrile	Dioxins & Furans
Di(2-ethylhexyl)phthalate	Asbestos
Glycol Ethers	Fine Mineral Fibers
Toluene-2,4-diisocyanate	BaP
Toluene-2,6-diisocyanate	



**Figure 3-1**  
**Location of MATES II Sites**

### 3.2.1 Intersite Comparison Concentrations

Figure 3-2 compares mean concentrations for each site along with its 10<sup>th</sup> and 90<sup>th</sup> percentile confidence intervals for the important carcinogenic compounds measured.

The charts for 1,3-butadiene and benzene show that the spatial patterns for the two pollutants are highly correlated. That is, peaks for both pollutants occur at Compton and Huntington Park and minimum values occur at Anaheim, Fontana, Long Beach, Pico Rivera, and Rubidoux. These differences are statistically significant and understandable since 1,3 butadiene and benzene are both products of on-road vehicles.

The spatial pattern of carbon tetrachloride is flat, as concentrations over the ten sites vary by only ten percent. In addition, observed 1995 annual concentrations of carbon tetrachloride at Santa Barbara and Chula Vista, sites well outside the South Coast Air Basin, are 0.10 ppb (ARB, 1999). This is in the range of values observed at the ten sites shown here. This ~~implies~~ shows that ~~there any is no~~ local source influences of carbon tetrachloride have and that the observed concentrations are essentially been reduced to near background conditions.

Huntington Park and Compton have higher p-dichlorobenzene concentrations relative to the other eight sites. Ambient styrene concentrations at Anaheim are, on average, more than twice the levels measured at the other nine sites. This implies local sources of p-dichlorobenzene at Compton and Huntington Park, and of styrene at Anaheim. The large variability in the data at Huntington Park and Anaheim indicate that the source (or sources) are proximate to the monitor such that on some sampling days the wind directs the emissions to the monitor and on other days the emissions are directed away from the monitor. Further analyses identified three major sources of styrene upwind of the monitoring site at Anaheim.

Peak perchloroethylene concentrations occur at Burbank and minimum concentrations occur at Fontana, Long Beach, Pico Rivera, and Rubidoux. Trichloroethylene concentrations at the Los Angeles site are two to six times greater than the concentrations measured at the other nine sites. Trichloroethylene concentrations at Anaheim are also elevated relative to Burbank, Compton, Fontana, Huntington Park, Long Beach, Pico Rivera, and Rubidoux. These differences are statistically significant at the 90 percent confidence level.

Formaldehyde and acetaldehyde are both primarily directly emitted and secondarily formed via chemical reactions in the atmosphere, with the principal source of formaldehyde, acetaldehyde, and their precursors being on-road mobile sources. The spatial pattern for formaldehyde and acetaldehyde is similar, which is reasonable given that both pollutants have common sources. Concentrations over the network vary by a factor of two. The downwind sites of Pico Rivera and Rubidoux have relatively high concentrations, which is also reasonable given that formaldehyde and acetaldehyde are secondarily formed.

The pollutants of acetone, methyl ethyl ketone, hexavalent chromium, nickel and selenium each

vary over the network by a factor of two to three. Burbank has higher methyl ethyl ketone concentrations than the sites of Los Angeles, Fontana, Huntington Park, Long Beach, and Wilmington; hexavalent chromium is highest at Rubidoux, Compton, Huntington Park, and Burbank; nickel is highest at Huntington Park and Pico Rivera; and selenium is highest at Huntington Park.

Elemental carbon is included in Figure 3-2 since it is treated as a surrogate to diesel particulate matter. Lead and elemental carbon exhibit somewhat similar spatial patterns. Huntington Park and Pico Rivera have the highest concentrations for both pollutants, while Anaheim and Long Beach have the lowest concentrations

### **3.2.2 Intersite Comparison Cancer Risk - Uncertainty in Risk Assessment**

There are inherent uncertainties in risk assessment with regard to the identification of compounds as causing cancer or other health effects in humans, the cancer potencies and Reference Exposure Levels (RELs) of compounds, and the exposure that individuals receive. It is common practice to use conservative (health protective) assumptions with respect to uncertain parameters. The uncertainties and conservative assumptions must be considered when evaluating the results of risk assessments. The following summarizes major areas of uncertainty and the assumptions used to address them.

Since the potential health effects of contaminants are commonly identified based on animal studies, there is uncertainty in the application of these findings to humans. In addition, for many compounds it is uncertain whether the health effects observed at higher exposure levels in the laboratory or in occupational settings will occur at lower environmental exposure levels. In order to ensure that potential health impacts are not underestimated, it is commonly assumed that effects seen in animals or at high exposure levels could potentially occur in humans following low-level environmental exposure (albeit at a lower rate), unless there is specific evidence to the contrary.

The estimation of cancer potencies and RELs is another major area of uncertainty. Estimates of potencies and RELs are derived from experimental animal studies or from epidemiological studies of exposed workers or other populations. Uncertainty arises from the application of potency or REL values derived from this data to the general human population.

With regard to cancer potencies, if the potency estimate is derived from animal studies, it is common practice by regulatory agencies to use an upper bound estimate of the potency of a compound in order to ensure that risks to humans from a given exposure are not understated. Similarly, the RELs developed by regulatory agencies commonly incorporate safety factors to ensure that they are health protective.

Uncertainty also exists in exposure estimates that are used to estimate risks. The risk assessment

procedures used by the District assume that there is no difference between indoor and outdoor pollutant concentrations although such differences may exist. The procedures also assess exposures as though individuals residing in the vicinity of a source remain in this location for a lifetime of 70 years. A different set of exposure assumptions may lead to lower exposure estimates and consequently lower risk estimates.

There is further debate as to the appropriate levels of risk ascribed to diesel particulates. CalEPA, in recommending a cancer risk level of 300 in a million per microgram per cubic meter ( $\mu\text{g}/\text{m}^3$ ) of diesel particulates, considered evidence which suggested diesel risks as low as 150 in a million to as high as ~~1,500~~2,400 in a million per  $\mu\text{g}/\text{m}^3$ . The USEPA has not yet declared diesel particulates as a toxic air contaminant. Thus, the selection of a risk factor for diesel particulates can have a substantial effect in assessing overall risks; however, ~~it~~even using the lowest bound of the CalEPA-recommended risk factor (150 in a million) does not change diesel's domination in the overall risks. For purposes of this study, and to be consistent with the approaches used for other toxic pollutants, the CalEPA recommended value of 300 in a million per  $\mu\text{g}/\text{m}^3$  is used.

Despite its inherent uncertainties, risk assessment remains the most useful tool available for estimating the potential health risks due to low-level environmental exposures measuring progress, and comparing between various locations. Several agencies are undertaking efforts to further refine the risk assessment process.

### 3.2.3 Intersite Comparison Cancer Risk

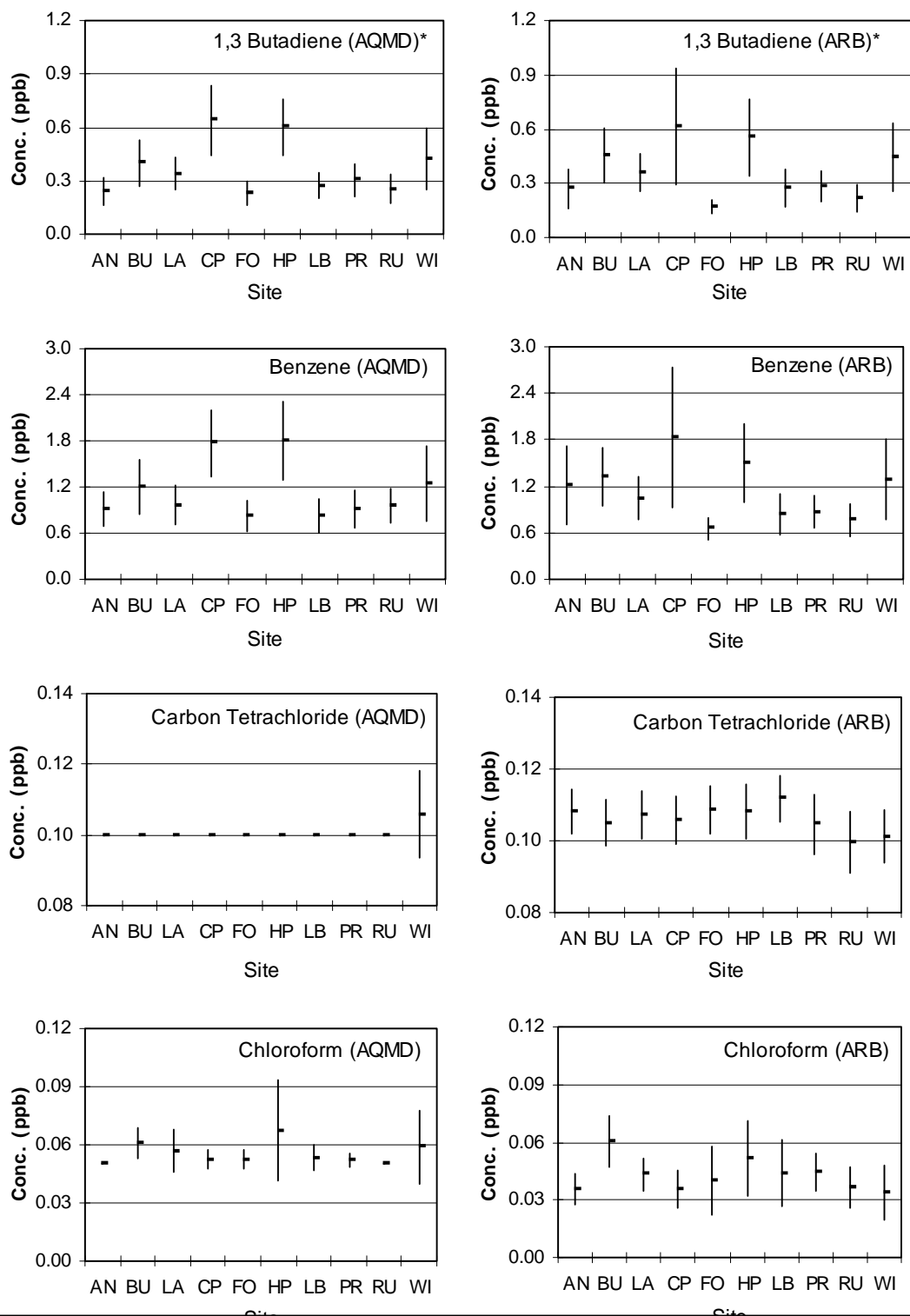
The total carcinogenic risks for the ten fixed sites are shown in Figure 3-3 (top chart). Two other charts are shown in the figure: one shows the risks from pollutants associated with mobile sources (middle chart, excluding diesel) and the other shows the risks from pollutants associated with stationary sources (bottom chart). Cancer risks are itemized by six key TACs (benzene, 1,3-butadiene, hexavalent chromium, carbon tetrachloride, perchloroethylene, and para-dichlorobenzene) and four lumped categories. The lumped category labeled "Carbonyls" consists of formaldehyde and acetaldehyde; the category labeled "Other VOCs" consists of chloroform, ethylene dibromide, ethylene dichloride, methylene chloride, and trichloroethylene; the category called "Other PM" consists of arsenic, beryllium, cadmium, nickel, lead, and selenium; and lastly the category named "PAHs" consists of benzo(a)pyrene, benzo(b)fluoranthene, benzo(k)fluoranthene, dibenzo(a,h)anthracene, and ideno(1,2,3-cd)pyrene.

As shown in the top part of Figure 3-3, cancer risks over the network vary by nearly a factor of 1.7, ranging from a low of about 310 in one million at Fontana, to a high of about 530 in one million. The higher levels are about the same at Burbank, Huntington Park and Compton. Note that 1,3 butadiene, benzene, and the carbonyls contribute 57 to 69 percent of the risk and as stated earlier the principle source of these toxics is the on-road motor vehicles. Also the risks from mobile sources vary by a factor of 2.0, whereas those from stationary sources vary by a factor of only 1.5. As seen in the bottom of Figure 3-3, the risks from stationary sources are

fairly uniform throughout the MATES II network. In other words, most of the spatial variability observed in the monitoring network is from 1,3 butadiene, benzene, formaldehyde, and acetaldehyde, -pollutants associated with mobile sources.

Figure 3-4 shows total carcinogenic risk including the diesel particulate contribution. The assumed unit risk factor for diesel particulate is  $3.0 \times 10^{-4} (\mu\text{g}/\text{m}^3)^{-1}$ . Research by Gray (1986), ~~which was -and-~~ used by ARB (1998) in their identification of diesel exhaust as a toxic air contaminant, is used here to estimate diesel particulate concentrations from the elemental carbon concentrations. Gray (1986) showed that approximately 67 percent of the fine elemental carbon mass in the Los Angeles atmosphere comes from diesel engine emissions, and that of all diesel exhaust particles emitted, elemental carbon averaged about 64 percent of this total. Therefore, diesel particulate concentrations are estimated by multiplying the elemental carbon concentrations by 1.04 ( $67\%/64\% = 1.04$ ). It should be noted that elemental carbon concentrations were not measured at Compton and Wilmington (due to physical limitations) so Figure 3-4 only shows the total cancer risks at eight of the ten fixed sites. In the figure, "Other" refers to the total contribution from the carbon tetrachloride, para-dichlorobenzene, perchloroethylene, other VOCs, hexavalent chromium, other PM, and PAHs.

The addition of diesel particulate toxicity dramatically increases carcinogenic risk. Risks range from a low of about 1120 in one million at Anaheim and Long Beach, to a high of about 1740 in one million. Those sites with the highest measured risk levels, Huntington Park, Pico Rivera, Los Angeles, and Burbank, are indicative of the urban core area surrounding Downtown Los Angeles. Diesel particulate, 1,3 butadiene, and benzene (all mobile source related) contribute 87 to 91 percent of the risk.



AN – Anaheim	BU – Burbank	LA – Los Angeles	CP – Compton	FO – Fontana
HP – Huntington Park	LB – Long Beach	PR – Pico Rivera	RU – Rubidoux	WI –

**Figure 3-2. Comparison of Concentrations Among MATES-II Sites.**

Tick mark represents the mean and the bar represents the 90 percent confidence interval about the mean.

\* (AQMD) and (ARB) refer to laboratories conducting analysis.



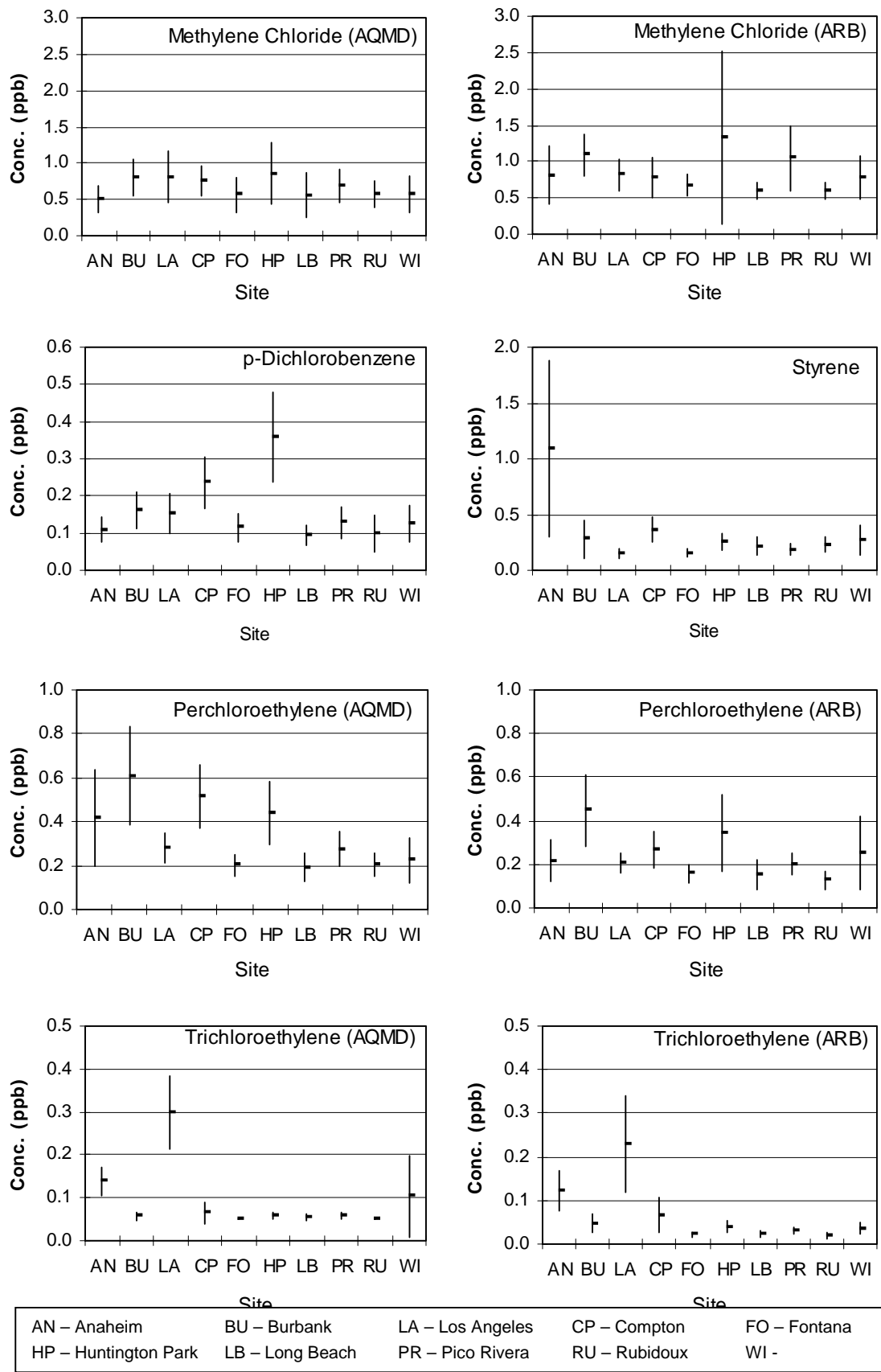
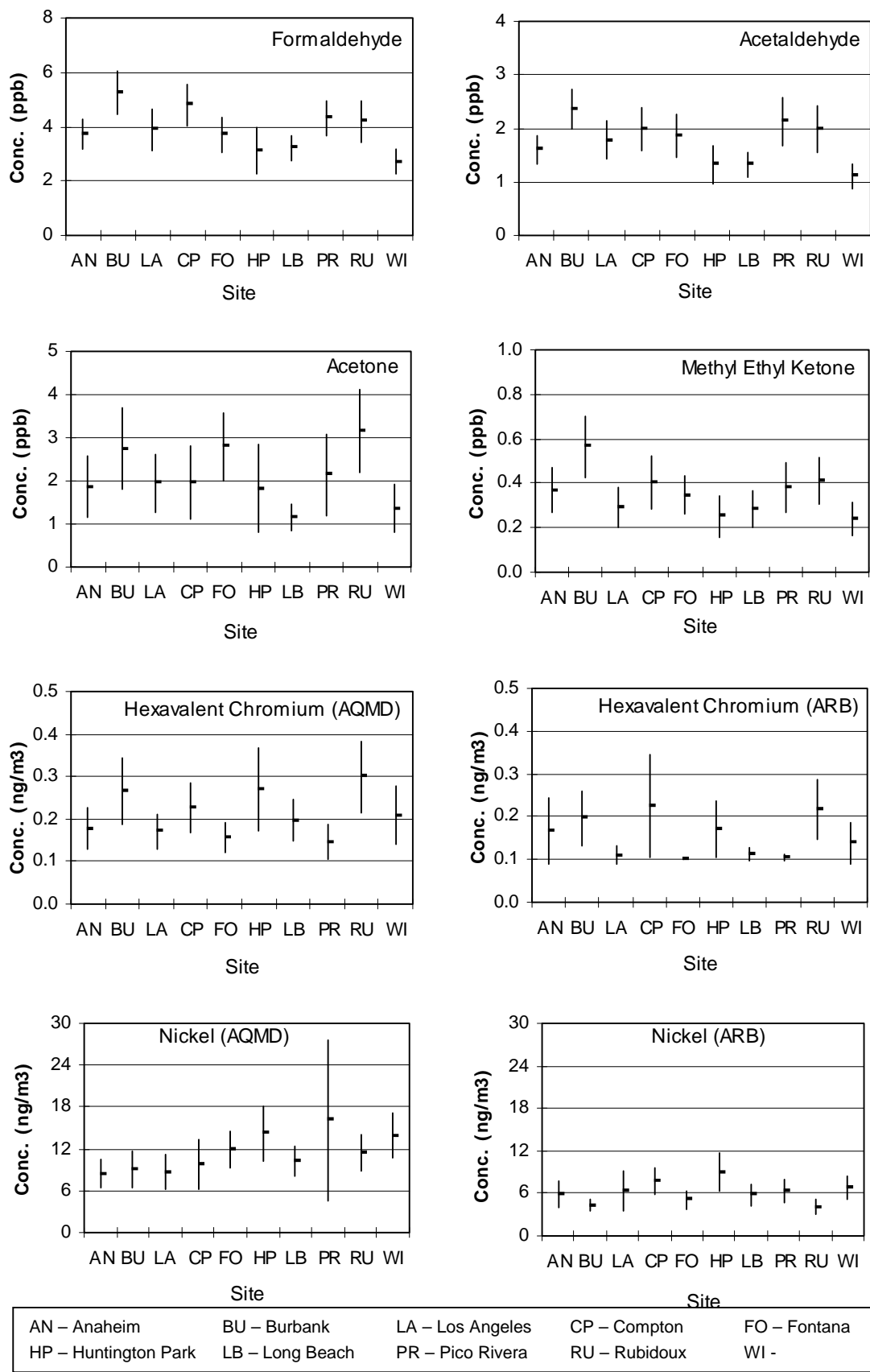
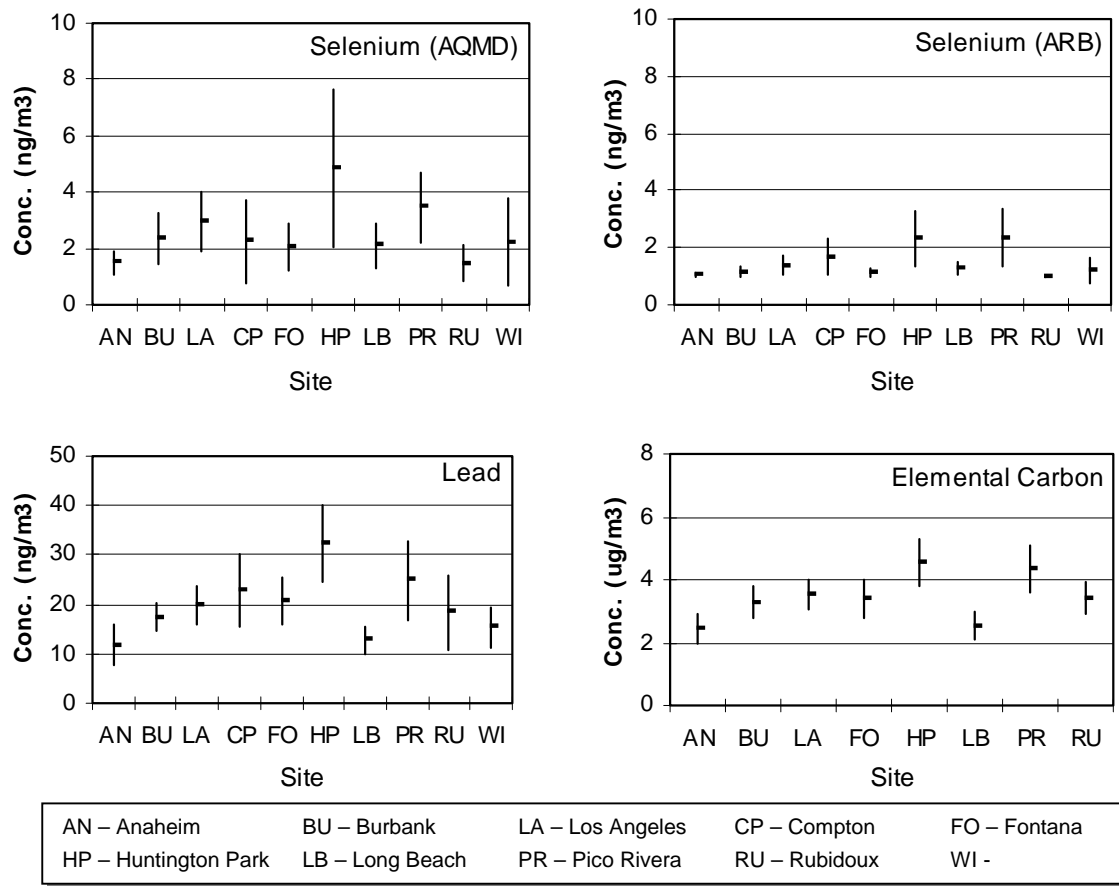


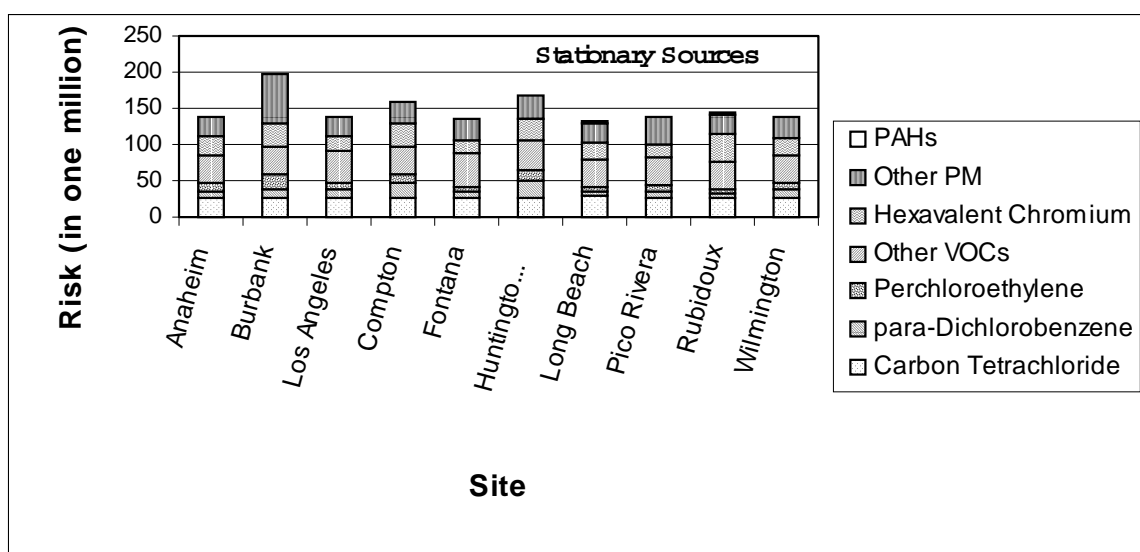
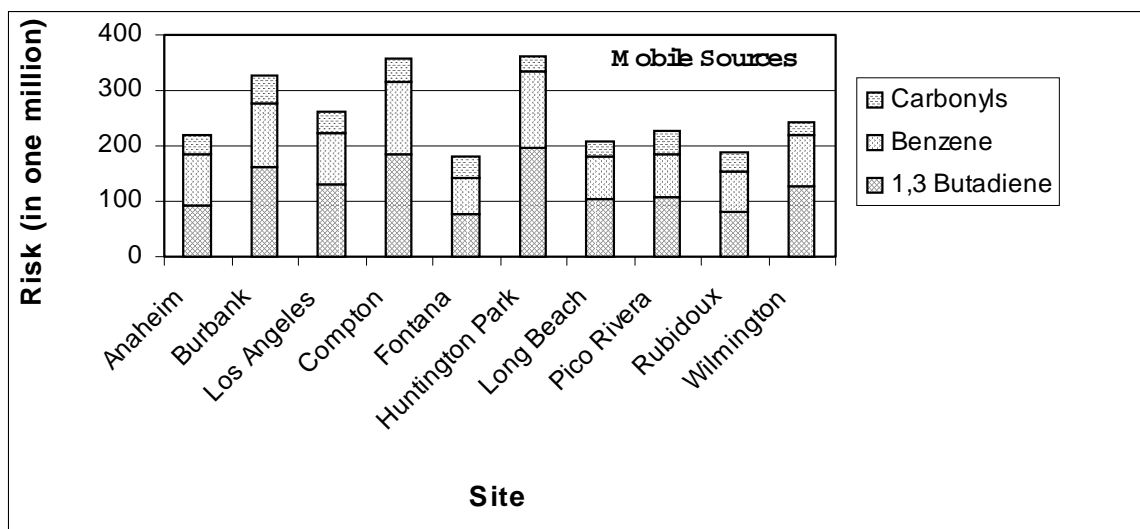
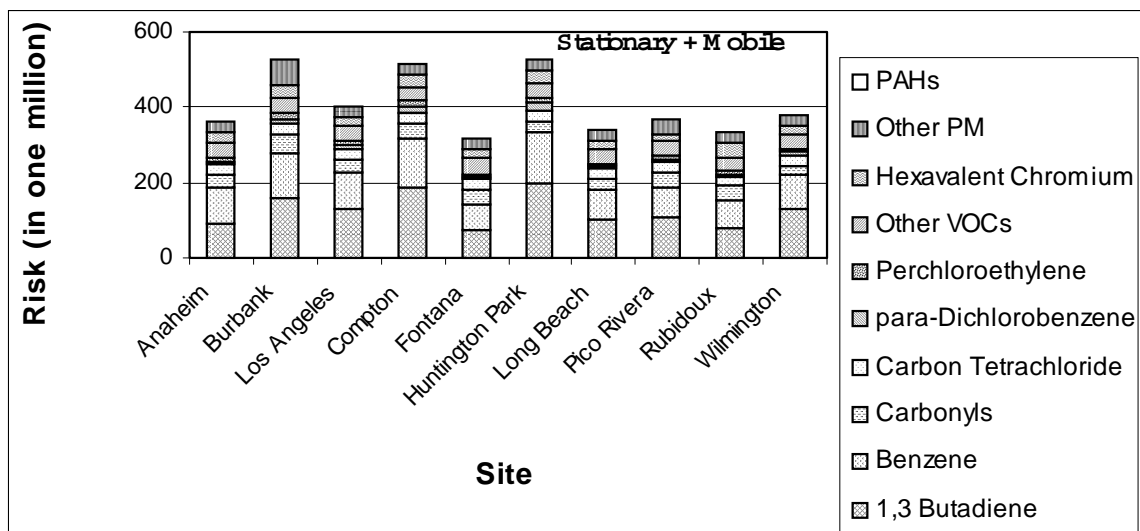
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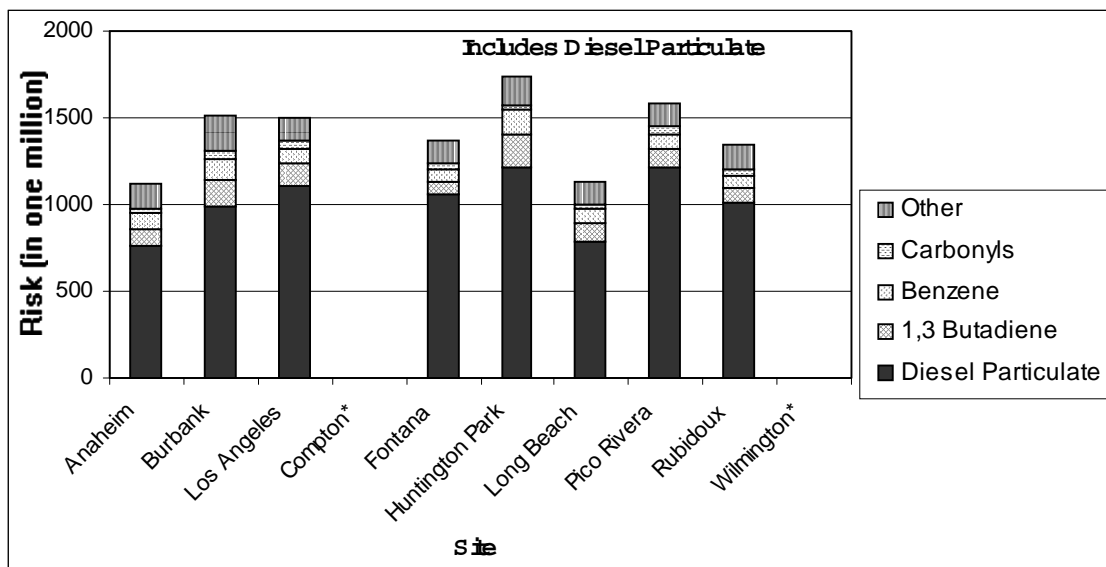
**Figure 3-2. Continued**



**Figure 3-2. Concluded**



**Figure 3-3. Cancer Risks at the MATES-II Fixed Sites.**  
 Risks are shown for all sources (top), mobile sources (middle),  
 and stationary sources (bottom), all excluding diesel toxicity.



**Figure 3-4. Cancer risks including diesel particulate toxicity at the MATES II fixed sites.**

\*No elemental carbon measured at these sites

### 3.2.4 Seasonal Variability

Monthly network mean concentrations and their 10<sup>th</sup> and 90<sup>th</sup> percent two-tailed confidence intervals are shown in Figure 3-5 for 18 of the measured pollutants.

Not all the pollutants exhibit a seasonal pattern but those that do follow two discernible patterns. Benzene, 1,3 butadiene, methylene chloride, perchloroethylene, methyl ethyl ketone, lead, and elemental carbon exhibit wintertime maximum and summertime minimum concentrations. This pattern is due to local seasonal meteorological conditions. Typically in late fall and winter, light winds result in reduced ventilation and late night and early morning surface inversions trap emissions from ground-level sources, such as on-road vehicles, and inhibit the vertical dispersion of pollutants, whereas, increased ventilation is typical of the summertime months. Stronger land breeze/sea breeze circulation and increased insulation results in increased wind speeds and increased vertical atmospheric dispersion and subsequently reduced ambient concentrations.

Formaldehyde, acetaldehyde, p-dichlorobenzene, and nickel exhibit a different seasonal pattern, which is a summer/fall maximum and a winter/spring minimum. Depending on the pollutant there are two potential causes for this pattern. Much of the measured formaldehyde and acetaldehyde are secondarily formed through photochemical reactions and thus the pattern follows cycle of increasing solar radiation. The peak is delayed since summertime is also the time of increased ventilation and vertical mixing which tends to reduce concentrations. However, late summer/early fall is a time of high insulation but somewhat reduced ventilation and vertical dispersion.

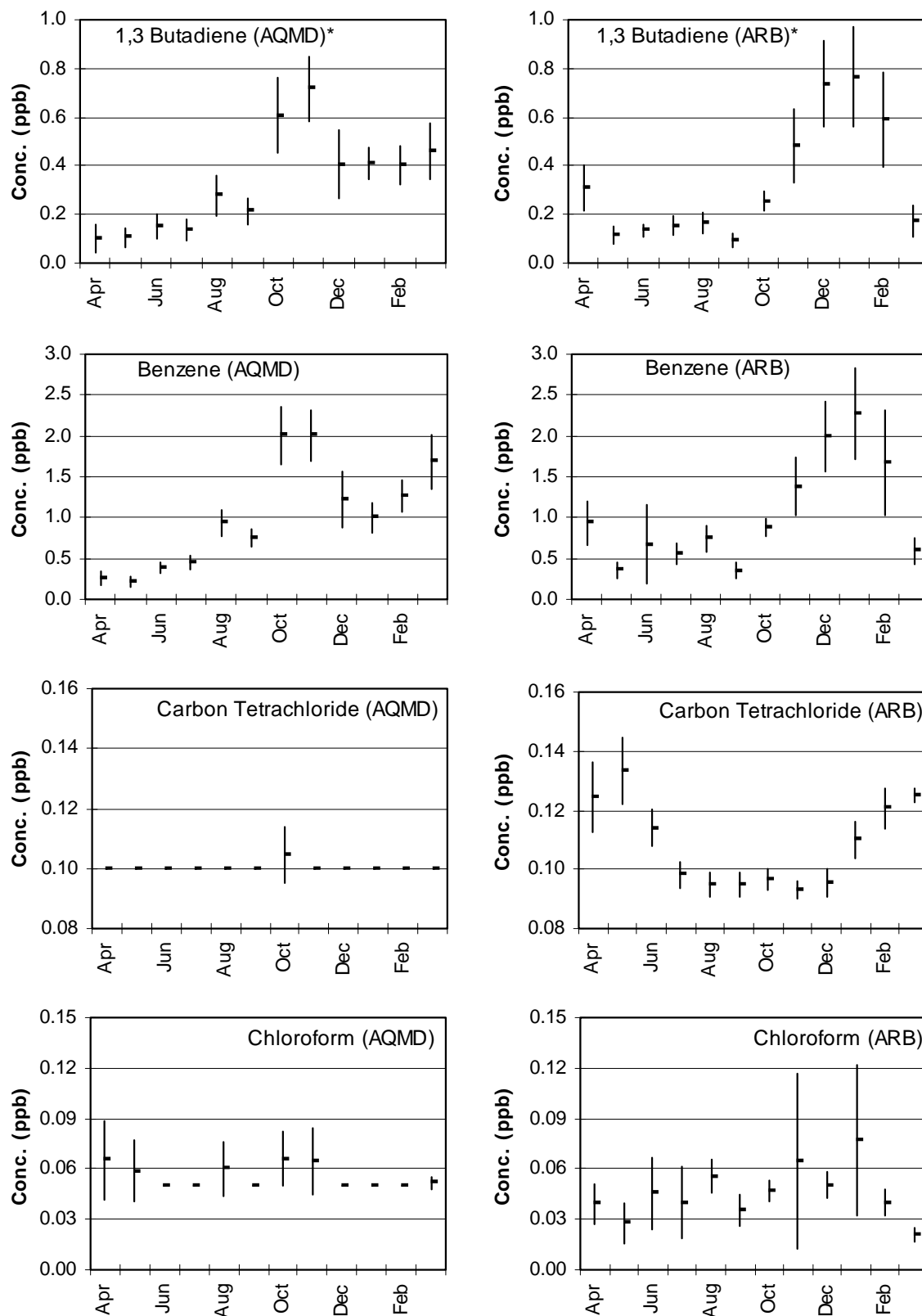
It is speculated that wind blown or mechanically disturbed crustal material is an important source of nickel. However, the original source of the nickel is man-made activities, which then are deposited on the surface of the soil. Wind blown dust is greatest during the dry season (i.e., summer and early fall) and least during the rainy season (winter and early spring), thus possibly explaining the seasonal pattern of the nickel concentrations.

Seasonal variations of the total cancer risks from the individual species are shown in Figures 3-6 and 3-7. The monthly values should be viewed as toxicity weighted concentrations and not as cancer risks since risks should be assessed based on long-term ambient exposure. A strong wintertime peak is shown in both figures. This is because much of the toxicity comes from pollutants that exhibit strong wintertime maximums, such as 1,3 butadiene, benzene, and diesel particulate.

In addition to monthly total carcinogenic risks, Figures 3-6 and 3-7 also show the monthly risks from pollutants associated with mobile sources and monthly risks associated with stationary sources. Note that the mobile source pollutants account for nearly all the monthly variability in the risk. Mobile source risk varies by a factor of about four, whereas stationary source risk varies by only a factor of 1.5. Therefore, most of the seasonal variability, observed in the monitoring network, is from the mobile source pollutants of 1,3 butadiene, benzene, formaldehyde, and acetaldehyde.

These seasonal results are intriguing, since meteorology would be expected to cause similar effects, whether stationary or mobile. There are several possible explanations for this observation:

- 1) While the three key mobile source toxic air contaminants exhibit similar seasonal trends, not all stationary source pollutants follow this pattern. The effect of different seasonalities for stationary sources is somewhat “cancelable” in an aggregate setting.
- 2) Operations of stationary sources may be more limited (e.g., daytime only; weekday only, etc.) such that differing results over a 24-hour period may occur even under comparable meteorology
- 3) Virtually all mobile sources are emitted within 5 meters of the surface, whereas some stationary sources have much greater emission release heights.



**Figure 3-5. Seasonal Variation of Toxic Compounds.**

Tick mark represents mean and the bars represent the 90 percent confidence interval about the mean.

\* (AQMD) and (ARB) refer to the laboratories performing the analyses.

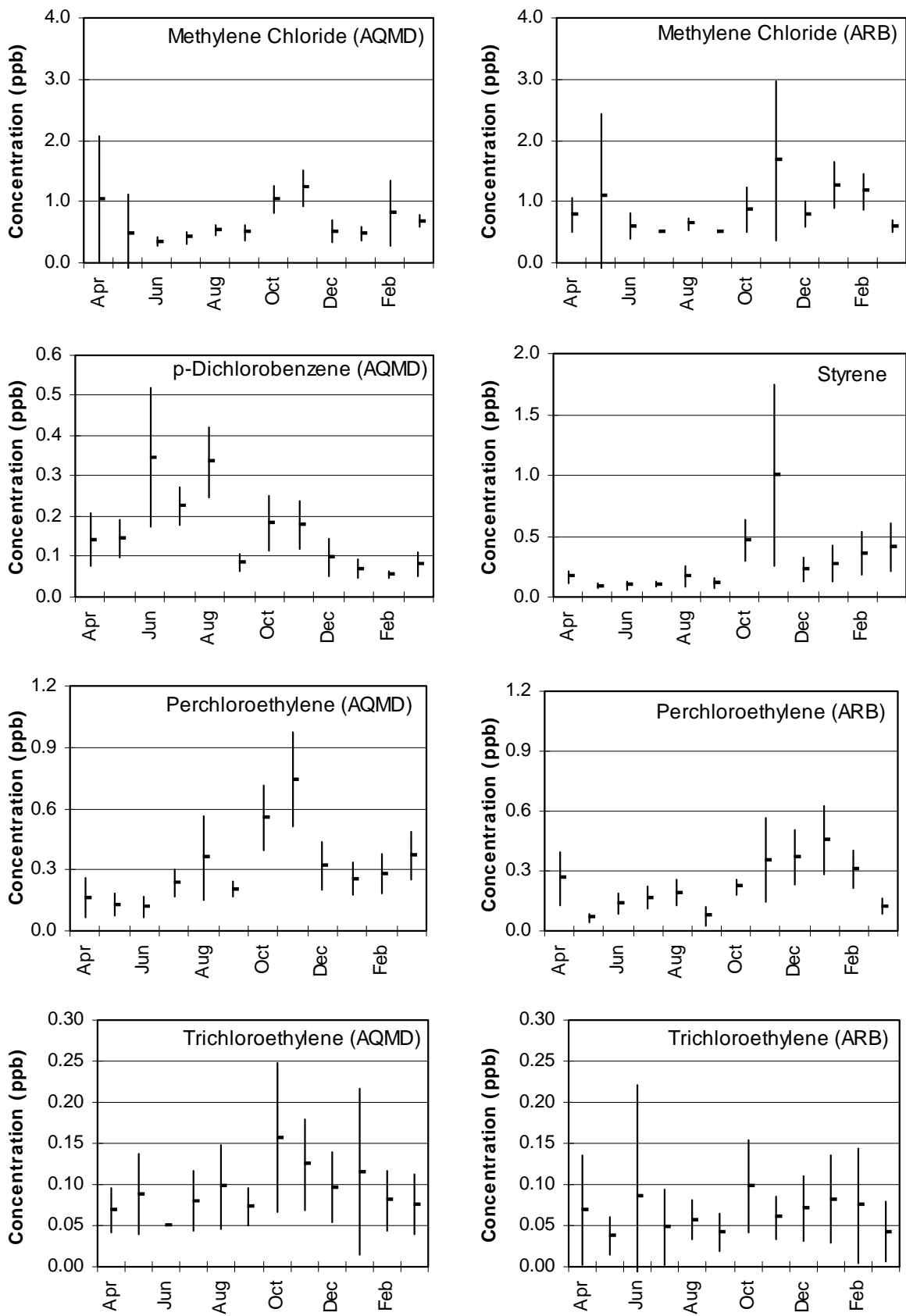
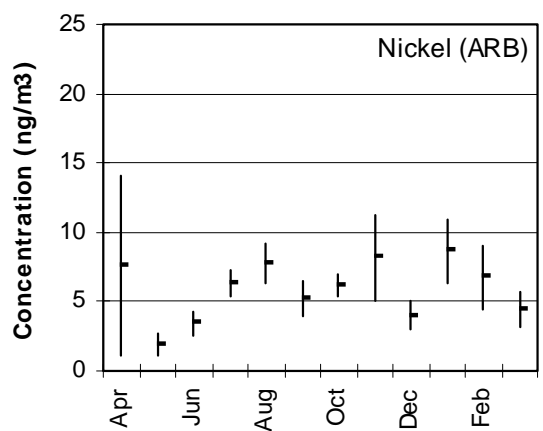
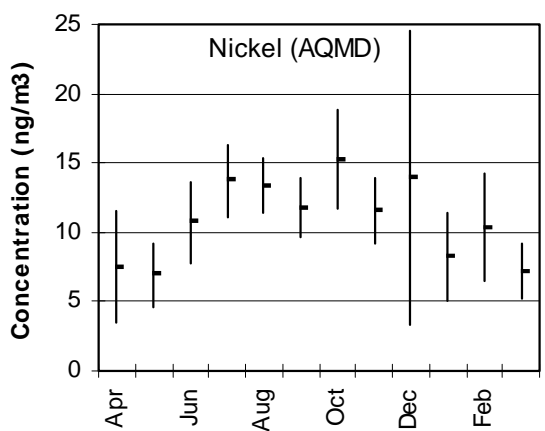
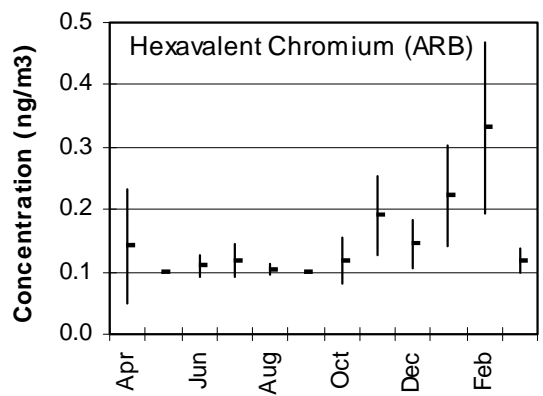
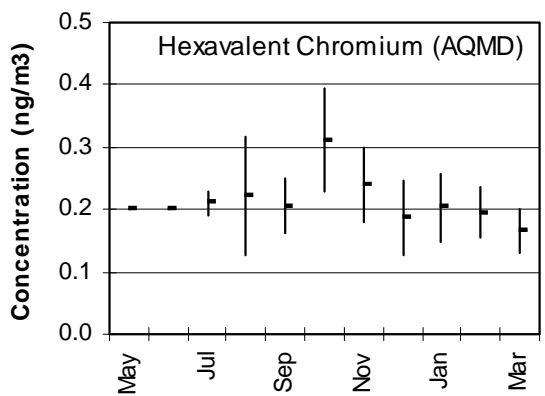
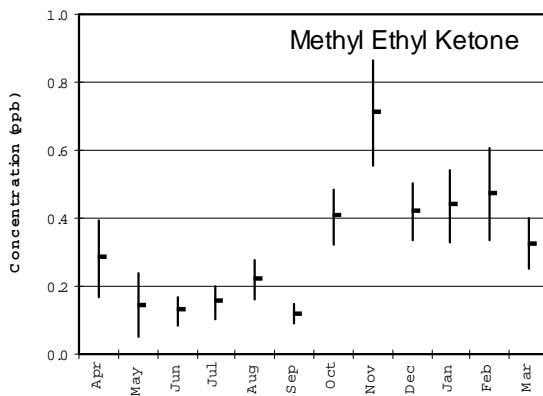
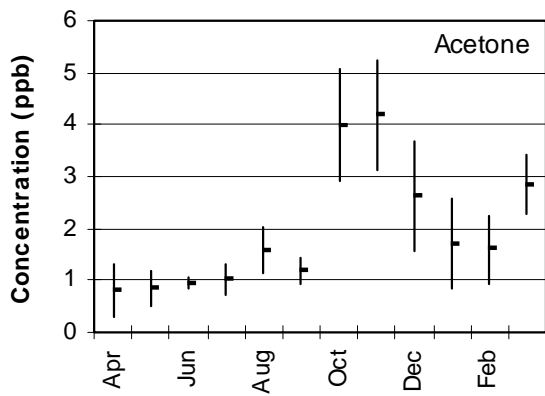
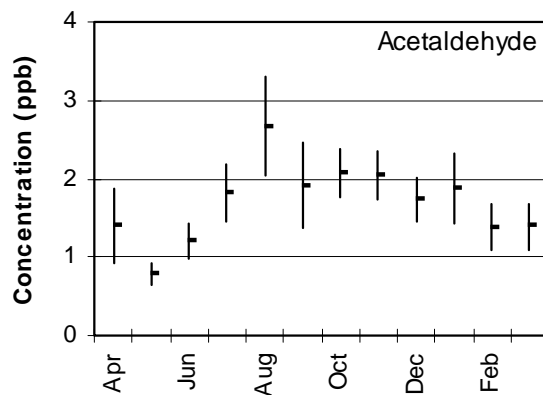
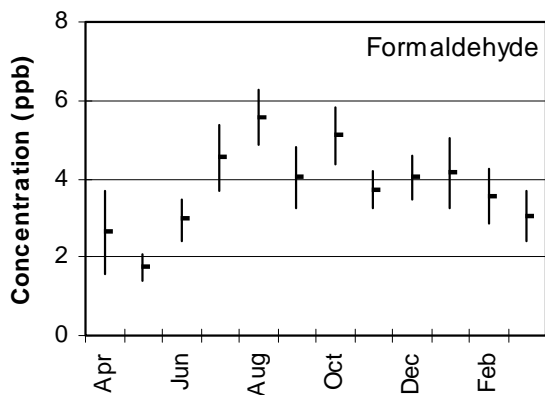
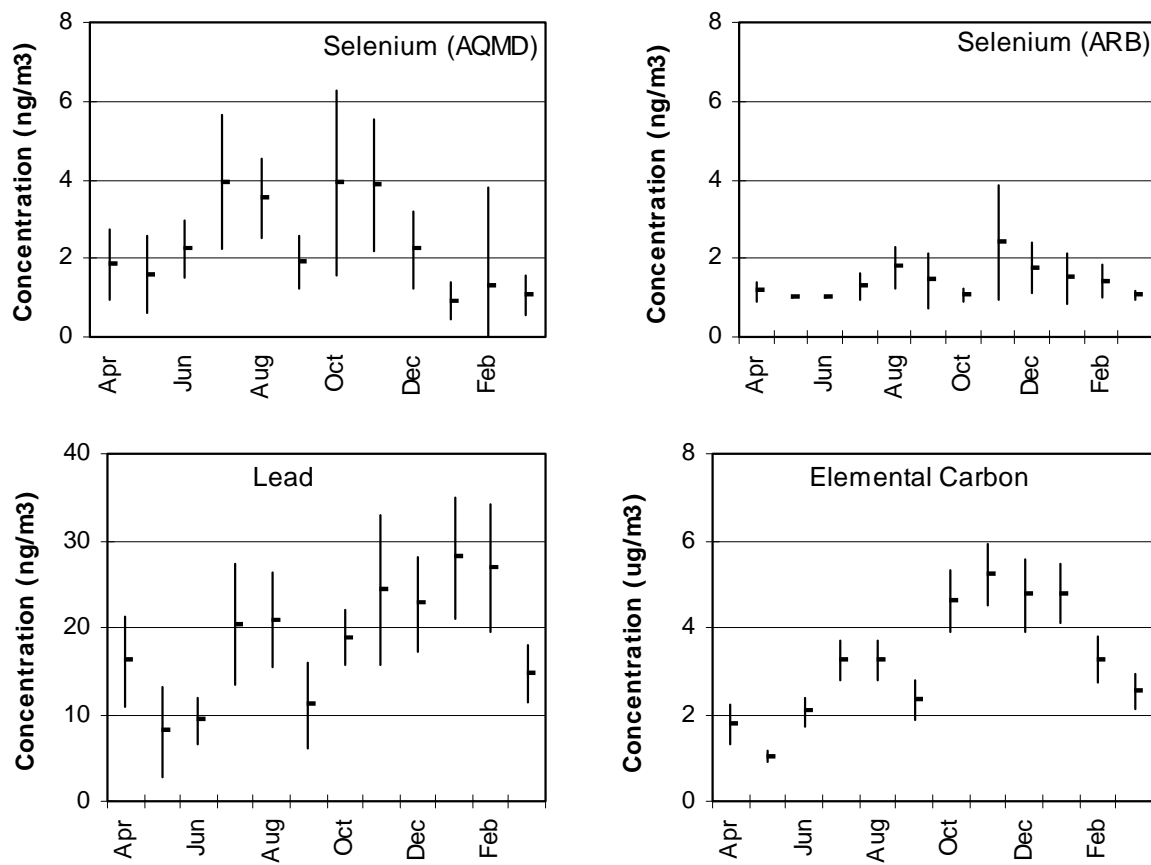


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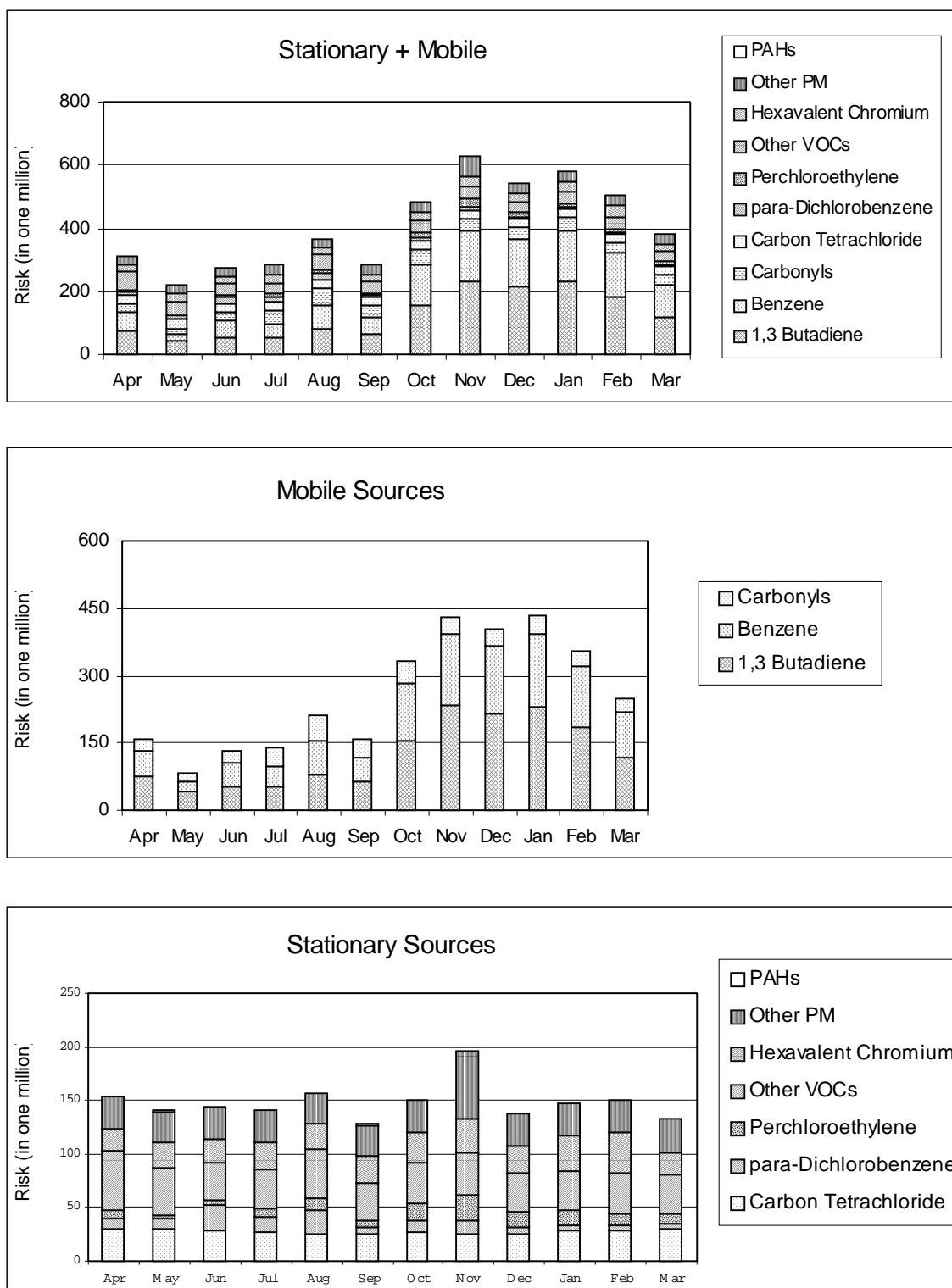




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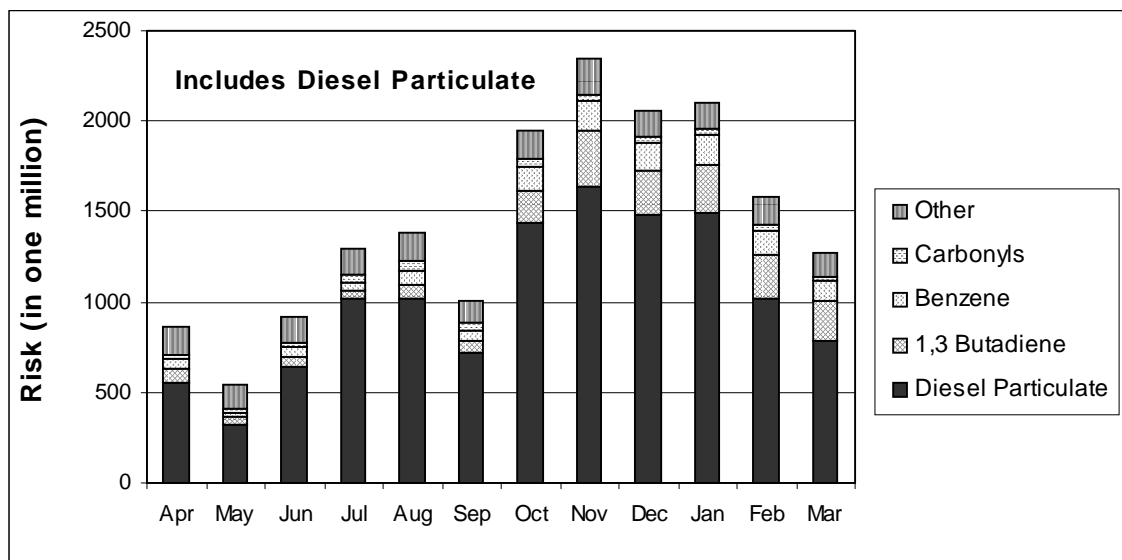


**Figure 3-5. Concluded.**



**Figure 3-6. Monthly variation in cancer risks\* excluding diesel particulate toxicity.**

\* Estimates to illustrate seasonal variation, not representative of lifetime exposure.

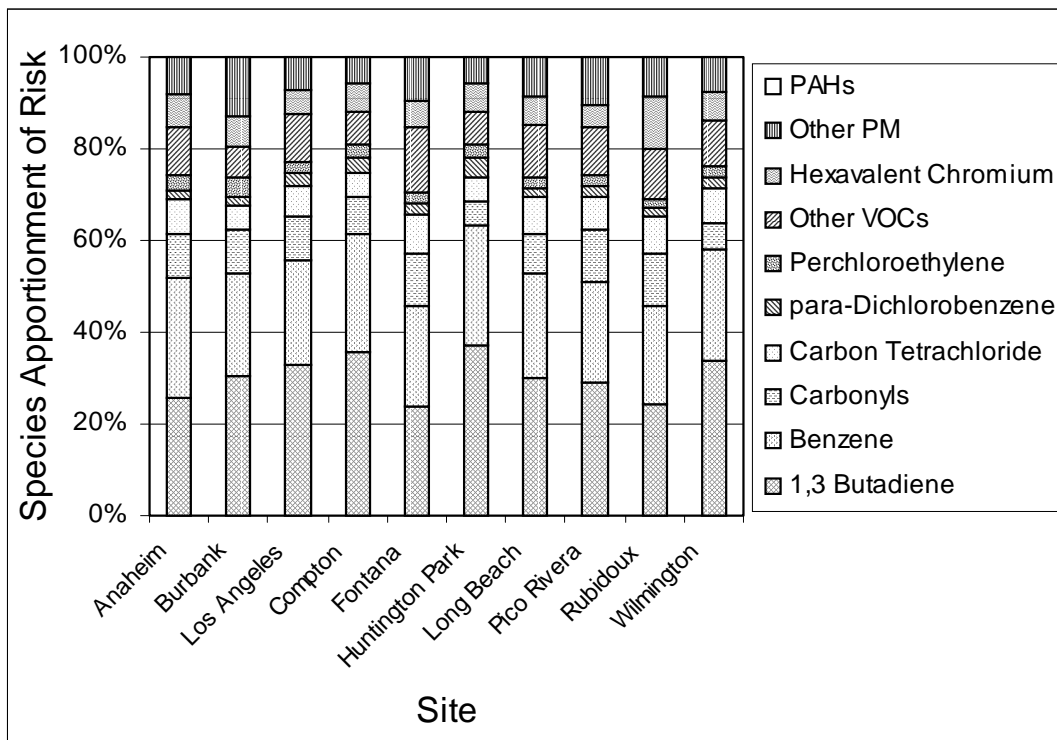


**Figure 3-7. Monthly variation in cancer risks\* including diesel particulate toxicity.**

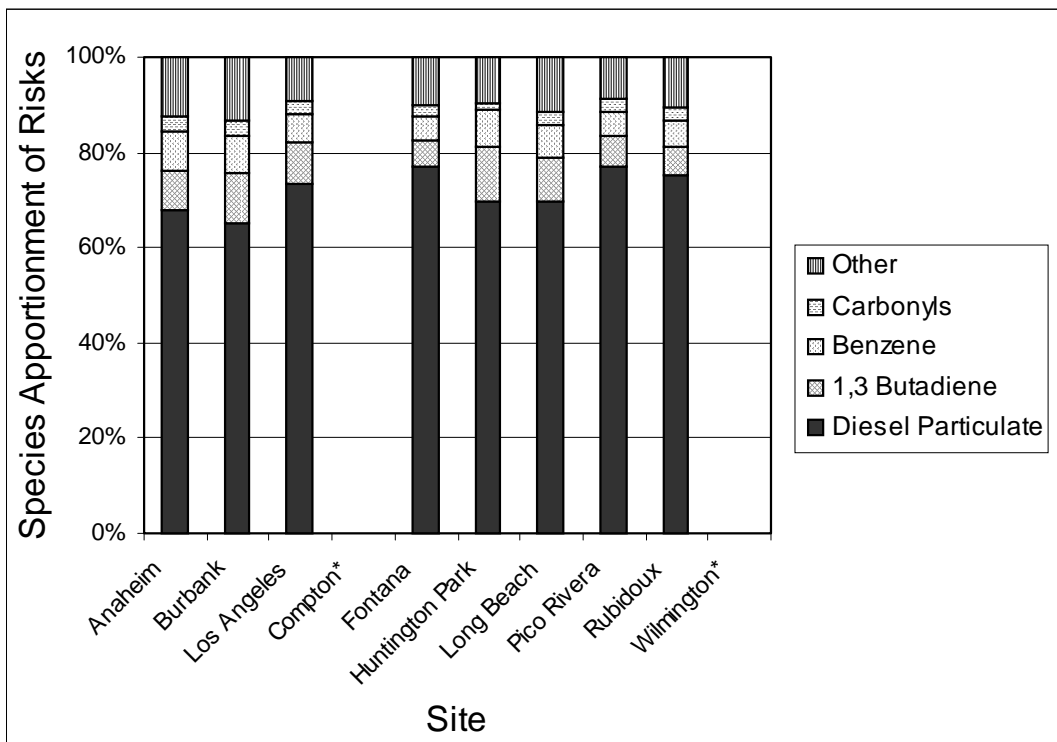
\* Estimates to illustrate seasonal variation, not representative of lifetime exposure.

### 3.2.5 Species and Source Apportionment

Figures 3-8 and 3-9 compare the percentage contribution of each toxic to the total risks, excluding and including the diesel particulate toxicity respectively. The charts are a convenient way of illustrating species apportionment and indirectly source apportionment. Note that at the ten fixed sites the mobile source pollutants of 1,3 butadiene, benzene, formaldehyde, and acetaldehyde contribute 57 to 64 percent of the cancer risks. With the inclusion of diesel particulate toxicity, the percent contribution to the risk increases to about 90 percent. There is relatively little variability of this apportionment breakdown throughout the Basin. However, the percent contribution from mobile source actually increases at those sites with higher risks.



**Figure 3-8. Species apportionment of cancer risks excluding diesel particulate toxicity.**



**Figure 3-9. Species apportionment of cancer risks including diesel particulate toxicity.**  
 \*No elemental carbon measured at these sites.